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The Authoritative Journal of Proctology, GASTROENTEROLOGY AND ALLIED SUBJECTS

AUGUST, 1959

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The Colon and Liver in Health and Disease

Intestinal Infarction Without Vascular Occlusion

Pneumatosis Cystoides Intestinorum Hominis

Intravenous Fat Emulsion

Newer Developments in Proctology

OFFICIAL PUBLICATION OF THE INTERNATIONAL ACADEMY OF PROCTOLOGY

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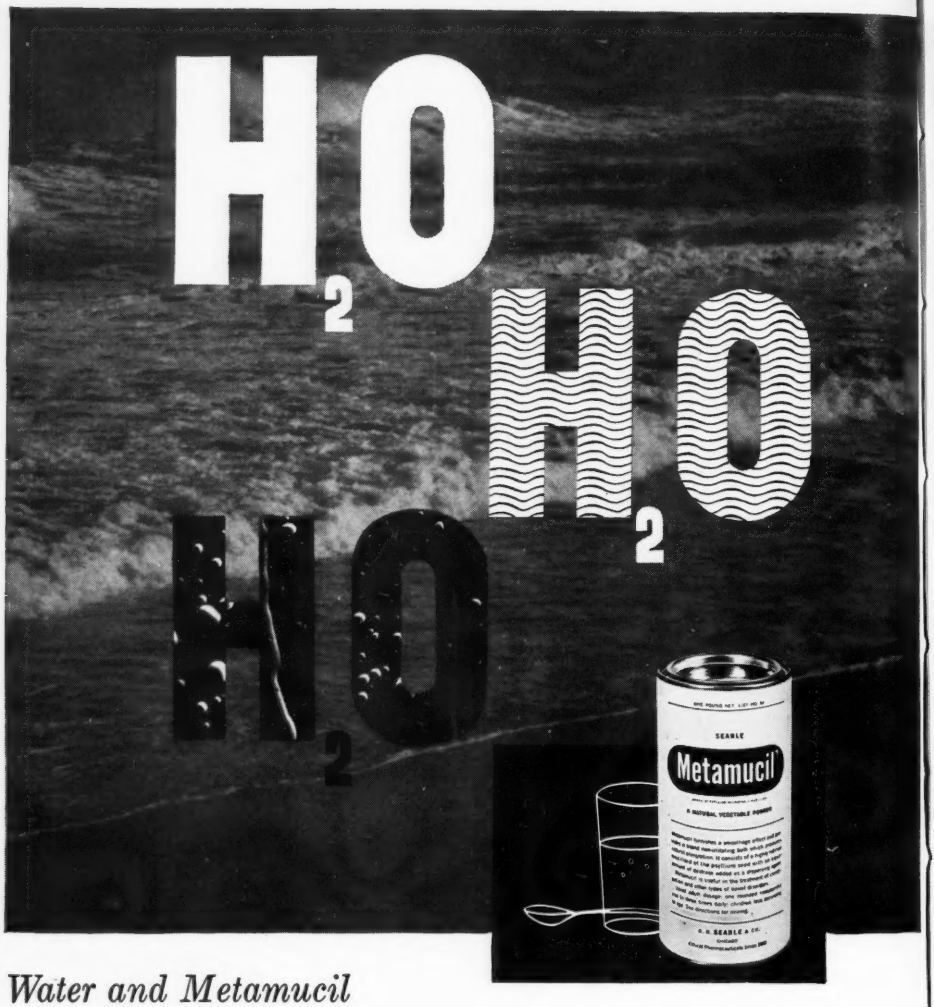

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LETTERS
To The Editor

This department is offered as an Open Forum for the discussion of topical medical issues. All letters must be signed. However, to protect the identity of writers who are invited to comment on controversial subjects, names will be omitted when requested.

Dear Sir:

An editorial in the June issue of the Journal, entitled "Prophylactic Proctology," states that a prophylactic cryptectomy should not be performed at the time of the removal of an abscess or fistula, but "at a later date." This seems to be contrary to previous teachings, as illustrated in "Ambulatory Proctology," and in teaching films that I have seen.

Further, there is the statement that "a properly balanced diet, etcetera, . . . will improve anyone's nutrition and will prevent any gastrointestinal disturbances" isn't this carrying it a little too far?

Sincerely,
L.J., M.D.
Chicago, Illinois

Dear L. J.:

The question of cryptectomy, as described in the editorial, deserves further explanation. Involved *crypts* associated *with a fistula should always be removed at the time of the fistulectomy. The editorial referred to cryptitis in other quadrants, unrelated to the existing acute pathology.*

As stated, a prophylactic cryptectomy of crypts in other quadrants, unrelated to the acute fistula, *is best performed at a later date. As stated, to do this surgery at the original time of the abscess and*

—Concluded on page 294

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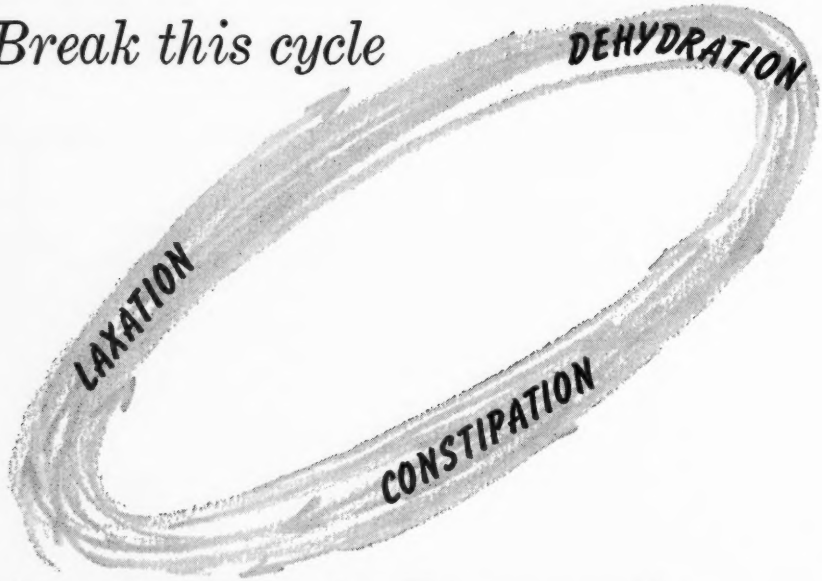

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Relationships Between the Colon and Liver in Health and Disease

S. S. LICHTMAN, M.D.*
New York, New York

The physiologic and clinical connections between the colon and liver are of interest to the physiologist, internist, surgeon and proctologist.

It is an oversimplification to state that the colon functions only as an emunctory organ dehydrating solid waste matter. Clinically, one deals with constipation, acute and chronic inflammatory and ulcerative diseases, parasitic involvement, diverticulitis, polyposis, abnormal pigmentation, disturbances in motility and malignant changes. The stool is examined for blood, ova, parasites and pathogenic bacteria.

The role played by the colon is far more important. In addition, the diseased colon may contribute to disease of the liver.

The Enterohepatic Cycles The bacterial flora of the colon plays a vital function in many activities, physiologic as well as clinical. Foremost, the colon and its bacteria are essential to various "enterohepatic cycles." The digestive ferments play no role in the transformations under consideration. These start in the caecum and onward.

The bile discharges bile pigment and salts, cholesterol, steroid and nonsteroid hormones, drugs and antibiotics into the gut. Upon reaching the caecum and onward, bacterial action brings about changes. Transformation products are resorbed, reprocessed or lost as surplus waste in the feces. Whipple, studying the synthesis of the bile acids by the liver, laid down the principle that the body would turn into a "pillar of bile salts" if a surplus was not eliminated in the stool.

The bile salts appear in the bile conjugated with taurine and glycine and in unconjugated form in the feces. Microbiologic activities do not start before the bile enters the caecum where the bile salts are deconjugated. Different strains of Clostridia and enterococci separate taurine and glycine from the bile salts.[1] The transformation products are mainly deoxycholic and keto bile acids. The Clostridia, bacilli alcaligenes fecalis and the coliform bacilli perform further changes. The intestinal flora exercises the dominating role on the rate

* Assistant Professor of Clinical Medicine, Cornell University Medical College.

Presented before the Eleventh Annual Meeting and Teaching Seminar, International Academy of Proctology, N. Y. C., April 8, 1959.

of fecal excretion of bile acids.

In the enterohepatic cycles, the transformation products are resorbed from the colon and circulated through the liver in the portal system. In the case of the bile acids, the liver reconverts deoxycholic acid into cholic acid. The keto bile acids resorbed from the colon are *hydrocholagogues* in contradistinction to cholic acid which is *choleretic*. The latter, conjugated, functions in response to meals, the former unrelated to meals.

Another enterohepatic cycle deals with the fate of the bile pigments. The coliform bacteria reduce bilirubin; stercobilinogen is converted into urobilinogen, resorbed and eliminated as such in the bile and urine. Suppression of bacterial action by bacteriostatic agents or hurried bowel movements causes bilirubin to appear in the stool unchanged. If bilirubin escapes transformation to urobilinogen, it does not appear in the urine. If this phenomenon goes unrecognized, it may be misconstrued that bile is excluded from the bowel by an obstructive lesion. The true situation is established by demonstration of unchanged bilirubin in the feces.

Synthesis of Vitamin K by Bacterial Action in the Gut Hypovitaminosis K may result from suppression of normal bacterial activities in the colon. The liver is able to appropriate vitamin K when supplied parenterally. The liver furnishes bile salts for solubilization of this fat soluble vitamin.

"Primary Biliary Constipation:" *A Specific Form of Hepatic Insufficiency* Constipation is a common complaint in middle-aged and elderly individuals. Often this is related to a clinical situation marked by spastic state of the colon or associated biliary tract disease. When associated with functional or organic disease of the liver or gallbladder, the designation "biliary constipation" implies that bile flow is impaired and constipation follows on this basis.[2] Bile normally contributes to the regulation of water balance in the colon. This irregularity of bile flow marks the *secondary* type of "biliary constipation.'

Our concern here is with the *primary* form of "biliary constipation." Its actual prevalence as a disease symptom is undetermined since it can be diagnosed only by clinical observation. It is defined as constipation associated with a partial insufficiency of bile salt formation by the liver. Routine testing of liver functions does not disclose other abnormalities. Present clinical facilities do not permit assay of liver bile for bile salt content.

Patients with primary biliary constipation state that conventional laxatives afford no relief; in fact they increase discomfort. In converse, relief is obtainable after they consume suitable amounts of natural bile salts or approximately three grams of desiccated whole bile containing natural bile salts, in three divided doses after meals. A favorable result is first realized about a week after medication is started and as long as it is continued. A week or so after it is discontinued the benefit tapers off. The administered natural bile salts spur hepatic synthesis of bile salts, i.e. choleresis. A normal enterohepatic circulation from gut to liver is reestablished. Constipation is relieved as long as there is a surplus in the bile salt pool. The same result is *not* obtainable with substitution of the hydrocholagogues or hydrocholeretics, i.e. dehydrocholic and ketocholanic acids.

Feeding of the recommended dosage

of desiccated whole bile or equivalent amounts of natural bile acids to normals or patients with constipation of a nonbilary type initiates disturbing diarrhea.

The only satisfactory method of establishing the diagnosis of primary biliary constipation is by testing the therapeutic response to a trial course of desiccated bile or equivalent natural bile salts. Unfortunately, the desiccated whole bile preparations are not standardized for bile salt content, so that the dosage equivalent in pure natural bile salts is not available. The proposed dose of three grams of desiccated whole bile appears to approximate the requisite daily dosage.

This medication is not to be confused with preparations containing antispasmodics, laxatives and minute amounts of bile salt derivatives.

Interference With The Enterohepatic Cycles Other cycles deal with steroid and nonsteroid hormones which appear in the bile. Some are excreted in inactivated state and eliminated in the stool as such. Interruption of cycles occurs in patients with occlusive jaundice. Bile salts, cholesterol and hormones are retained in the blood, lymph and tissues. Endocrine disturbances, pruritus, xanthelasmata, metorrhagia, hypoprothrombinemia and other changes follow.

Portal Hypertension and Rectal and Colonic Varices The colon and rectum become involved occasionally in the course of chronic liver disease. There are no reliable figures on the relative incidence of rectal varices in patients with hepatic cirrhosis in comparison with normals of corresponding ages. One aspect of the subject is provocative: Is excision of bleeding piles in cirrhotics contributory to vicarious bleeding from oesophageal bleeding? Is rectal bleeding as effective in reducing portal hypertension as epistaxis is in lowering arterial hypertension?

An indirect method of measuring portal tension was introduced by Newman and Cohen.[3] Ether is introduced per rectum and the circulation time between the superior and medial hemorrhoidal veins and the lung measured. The accuracy of the method has been challenged.[4] This is understandable since portovenous shunts introduce variables. Another method depends on resorption of acetylene gas from the duodenum.[5] This method is rated superior in comparison by some.[6]

Severe congestion and varices have been identified in the colon as causes of severe hemorrhage in patients with hepatic cirrhosis. This occurred in 4 of 47 patients studied by Enquist and Gliedman at the State University Medical Center in New York.[7] Fatal bleeding from a varix located in the colon has come to our attention in a case of decompensated portal cirrhosis.

Symptoms of Liver Disease Derived from Disturbance in the Colon Connections between the liver and brain are well documented. For example, hepatic coma is a common terminal event. Exogenous as well as endogenous factors contribute to its onset. A high protein diet and some therapeutic agents containing nitrogen favor access of ammonia to the systemic circulation. A principal source of ammonia is a high protein diet, especially rich in meat. "Meat intoxication" in Eck fistula animals commonly followed use of meat and meat extractives. A rough correlation between arterial blood ammonia concentration and portosystemic encephalopathy has recently received wide

attention. Measures designed to reduce ammoniagenic coma are: fasting, low protein diet, the use of broad spectrum antibiotics, purgatives and enemata. The bacterial count in the colon is reduced by such measures. Certain strains of microorganisms in the colon, especially the colon bacillus, form ammonia from nitrogenous substances such as amino acids, polypeptides and urea.[8]

The bowel was stressed for many decades past as a potential source of toxic products. Purgatives and enemata were recommended for so-called "autointoxication." These measures were discredited and frowned upon because of indiscriminate resort to this type of treatment as a panacea. More recently, use of such measures as reduced protein intake and broad spectrum antibiotics, have been recommended for reversing and preventing ammoniagenic coma.

The hemorrhagic tendency of liver disease may originate in suppression or rapid change of the bacterial flora of the colon. Lack of dietary Vitamin K which fails of absorption when bile is excluded from the bowel, and failure of bacterial synthesis of Vitamin K also contributes to development of hypoprothrombinemia. Prolonged use of oral antibiotics and diarrhea accomplish the same result.

Hepatic Dysfunction and Disease Related to Disease of the Colon and Rectum In view of the intimate vascular relationship between the liver, colon and rectum, it is suprising that the liver is not more often seriously affected by diseases of the rectum and colon.[9] The detoxifying functions of the liver probably account for the relative immunity of the organ.

Liver damage is not infrequent in patients with nonspecific ileocolitis[10] and less frequent in ulcerative colitis.[11] The occurrence of hepatic cirrhosis in ulcerative colitis has been decribed.[12]

Significant hepatic disease encountered in fatal ulcerative colitis includes: fatty metamorphosis, toxic hepatitis, cholangiolitic hepatitis, sclerosing cholangitis, biliary cirrhosis. Homologous serum hepatitis may complicate ulcerative colitis.

Hepatic lesions are frequent in amebiasis. Pyogenic abscess of the liver and suppurative pyelophlebitis complicate inflammatory lesions of the colon and rectum.

Metastatic cancer of the liver result from involvement of contiguous organs or it is disseminated by portal vein from the common sites of malignant tumors in the alimentary tract.

It has been postulated as an attractive hypothesis that the portal "streamlines" segregate the blood currents. Distribution is visualized as lobar: the left lobe receives a disproportionate amount of blood from the superior mesenteric vein. This arrangement would channel the distribution of liver abscess or cancer metastasis. Clinical experiments with radioactive sodium do not bear out the theory that there is a marked assymmetry in the distribution of the portal blood between the right and left hepatic lobes. It cannot be postulated with any degree of certainty that a patient with carcinoma in a certain portion of the gut is more likely to develop metastasis in one lobe of the liver than in another.[9]

Summary

There are definite anatomic, physiologic and clinical connections between the colon, rectum and liver.

Enterohepatic cycles exist. These serve to regulate the loss of surplus waste, reprocessing, and utilization of components of the bile such as bilirubin, bile salts, cholesterol, steroid, nonsteroid hormones, and drugs and antibiotics.

A specific form of "biliary constipation" is described. The liver forms subnormal amounts of bile salts. The liver is insufficient in this respect alone. Tests show no other failings. Diagnosis can be made by clinical observation alone. Accuracy of diagnosis is confirmed by response to therapeutic trial of feeding adequate amounts of natural bile salts or dessicated whole bile. The relative frequency of this form of isolated hepatic insufficiency affecting the motility of the colon is unknown.

The microorganisms inhabiting the colon influence the enzymatic transformations of bilirubin, bile salts, cholesterol, hormones, Vitamin K and other substances in the large gut. Diarrhea and oral and parenteral broad spectrum antibiotics suppress these bacteria and modify the transformation processes in the colon with favorable and unfavorable consequences.

Portal hypertension associated with chronic liver disease may entail severe free bleeding in the colon from congestion and varicosities.

Ammonia is generated in the colon by bacterial action on nitrogenous substances. Access of excess ammonia to the liver and brain may be sharply curtailed by consumption of less protein, use of broad spectrum antibiotics, fasting, purgation and enemata, and ammoniagenic coma thus prevented. The microorganisms inhabiting the colon play vital roles in the enterohepatic cycles and in ammonia formation.

Hepatic dysfunction and organic liver disease have been observed in patients with inflammatory, ulcerative, parasitic and neoplastic disease of the colon.

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OBITUARY

James J. Morrow, M.D., 1898-1959

THE INTERNATIONAL ACADEMY of Proctology lost a most loyal and valued member of its House of Delegates in the sudden death of Doctor James J. Morrow of North Hollywood, California. He passed away in his sleep at home on the morning of June 11, 1959.

He was a proud native of Minnesota, graduating from the Medical School of The University of Minnesota in 1924. He served with distinction in both World Wars. In World War I, Doctor Morrow was one of the original founders of The American Legion at their historic organizational meeting in Paris, France on March 17, 1919. During World War II he served on the Staff of General Douglas MacArthur in the Pacific. He held the rank of Colonel in the Army Medical Reserve Corps.

For eighteen years Doctor Morrow was a prominent member of the Medical Profession in Austin, Minnesota.

After World War II, Dr. Morrow decided to forsake the cold Minnesota and live in California.

In the fifteen years we were privileged to know Doctor Morrow here in Southern California, he made a host of friends both among the laity and in the profession. He was the acknowledged leader in San Fernando Valley not only in medical circles, but also in its active community affairs. Doctor "Jim" served with great distinction as a former Chief of Staff of the rapidly expanding and ultra modern St. Joseph's Hospital in Burbank as well as the immediate Past-President of world renowned Hollywood Academy of Medicine.

Doctor Morrow was the recognized Dean of Surgery throughout the far flung reaches of the San Fernando Valley. His diagnostic opinion and his superlative surgical skill were in constant demand. He gave unselfishly of himself so that his fellow man might enjoy prolonged health and happiness. Doctor "Jim" was a Doctor's Doctor. He was an able and articulate exponent of Medicine's role in the present day world with its many complex problems.

Doctor James J. Morrow has made an indelible mark upon the hearts of all of us who were privileged to have shared in his friendship and to have worked beside him as he accomplished many great achievements for us here in Southern California.

The International Academy of Proctology extends its deepest sympathy and condolences to the bereaved Wife—Hazel and Family of Doctor James J. Morrow.

Intestinal Infarction Without Vascular Occlusion

SEYMOUR LEVINE, M.D.
Jersey City, New Jersey

Infarcts of the small intestine may be caused by superior mesenteric artery embolism or thrombosis, superior mesenteric vein thrombosis, or by mechanical compression of these vessels or their branches as in strangulated hernia, volvulus or intussusception. There remain a few cases of intestinal infarction without demonstrable vascular occlusion ("functional infarcts," "unexplained infarcts"). Although these cases are few, they warrant special attention because they may be amenable to non-surgical therapy.

A number of cases of this type have been reported in the European and Latin-American literature. There are a few references to this subject in the English language literature[1] and the statement of Boyd:[2] "Occasionally the signs of infarction may be present although no obstruction can be found in the mesenteric vessels." It is possible that this condition is not really rare, but goes unreported because of the negative nature of the important finding (absence of vascular occlusion) and because of the ever-present possibility that a small occlusion may have been overlooked. In addition, in some cases, cardiac disease with mural thrombus may have been present,[3] raising the possibility (however remote) of an overlooked embolus.

Most cases previously reported have been based on observations made at laparotomy.[4-17] The diagnosis of intestinal infarct was made by inspection and usually without histologic confirmation. The absence of mesenteric vascular occlusion was not usually demonstrated anatomically but was assumed either because the patient survived without intestinal resection or because the appearance of the intestine returned to normal

The experimental work reported in this paper was supported by a grant from the Intestinal Research Institute. Dr. S. V. De Marco kindly gave permission to publish the case report and provided the clinical data.

under the eyes of the surgeon. These observations are open to question because spontaneous recovery from intestinal infarction has been observed in cases where subsequent autopsy has revealed vascular occlusion.[18] Therefore, autopsy material[1, 2, 19] is essential to establish the existence of intestinal infarction without vascular occlusion.

The case reported here seems to be of particular importance for several reasons: a complete autopsy was performed; there was no cardiac pathology and no possible source of emboli; arteriosclerosis was minimal; the great extent of the infarct with involvement of jejunum, ileum and ascending colon ruled out the possibility of a small (overlooked) vascular obstruction, and most important, the superior mesenteric vessels were dissected and opened *in situ* before any organs were removed.

Report of Case The patient was a 64-year-old white female who was admitted on October 20, 1958. The present illness began two days before with vague abdominal pain, nausea, vomiting and diarrhea. On the morning of admission she had a soft bowel movement and shortly thereafter a generalized convulsion occurred.

Past history included thyroidectomy in 1920, and pan-hysterectomy and appendectomy in 1936 for uterine fibromyomas. During the last nine years there have been complaints of intermittent chest and abdominal pains and the blood pressure has varied from 126/82 to 198/110. X-rays in 1954 and 1956 showed diverticulosis and diverticulitis of the sigmoid colon. The patient was noted to be a sensitive, tense and nervous individual.

Physical examination revealed a blood pressure of 220/110 with temperature 100°F and pulse 108. The patient was nearly comatose. Heart sounds were weak and irregular. Lungs were negative. Abdomen was soft and not distended but tenderness was elicited in the epigastrium. After rectal examination, tarry material was noted on the glove. No abnormal neurological findings were noted. Laboratory studies revealed hematocrit 46%, white cell count 39,750 (84% polymorphonuclear leukocytes, 3% bands, 2% metamyelocytes, 7% lymphocytes, 4% monocytes), blood sugar 116 mg. %, blood urea nitrogen 45 mg. %. Urine contained 12-16 leukocytes and many erythrocytes per high power field.

Course. A few convulsive seizures occurred in the hospital. The systolic blood pressure dropped to 100 and within a few hours the patient went into shock and died nine hours after admission. The final clinical diagnosis was cerebrovascular accident due to hypertension.

Autopsy Findings The autopsy was performed by the author. On opening the abdomen, all the jejunal and ileal loops were found distended and discolored dark purple to black (Figure 1). Immediately, the superior mesenteric artery and vein and the portal vein were dissected *in situ*, opened and found

---

Presented before the Eleventh Annual Teaching Seminar, International Academy of Proctology, New York, New York, April 8, 1959, from the Department of Pathology, St. Francis Hospital, Jersey City 2, New Jersey.

The International Academy of Proctology announced its Eleventh Annual Teaching Seminar Certificate of Merit Award at the Annual Dinner Dance, Wednesday evening, April 8, 1959. The Certificate of Merit was awarded to Dr. Seymour Levine, for this research paper, "Intestinal Infarction Without Vascular Occlusion."

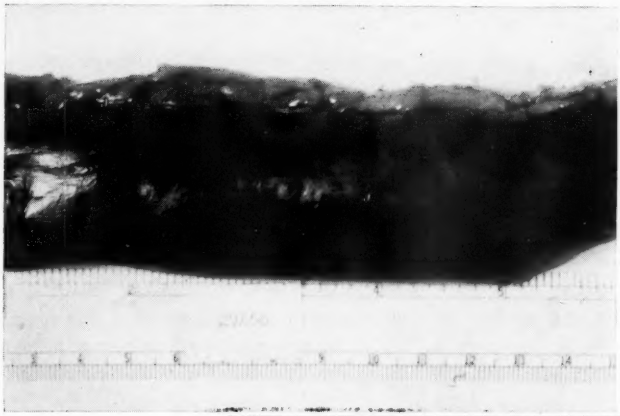

Figure 1. Portion of infarcted small intestine showing distention and discoloration.

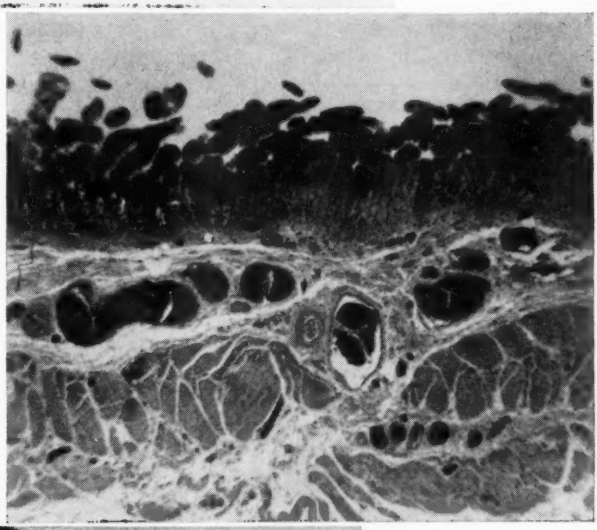

Figure 2. Hemorrhagic infarction of small intestine. Intense vascular congestion in all layers and hemorrhage in mucosa. Hematoxylin and eosin, X30.

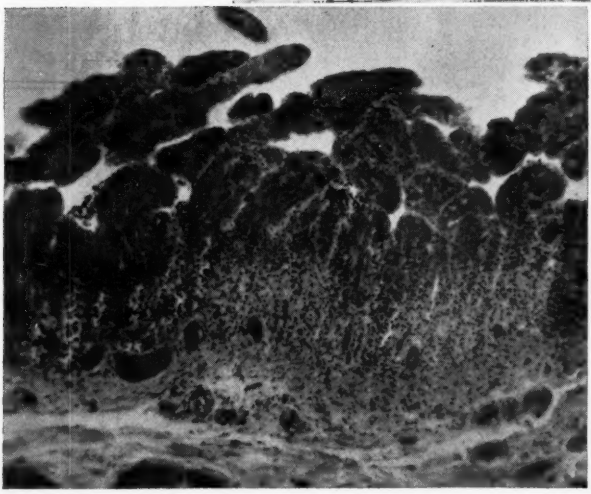

Figure 3. Necrosis of glands and hemorrhage in villi. Hematoxylin and eosin, X80.

patent. The artery showed minimal arteriosclerosis, but there was no narrowing, thrombosis or embolism. After removal of the viscera, the aorta was opened *in situ* and the patency of the superior mesenteric artery orifice was confirmed. Although all loops of jejunum and ileum were involved, the discoloration varied in degree in different areas. The discoloration extended to the cecum and ascending colon, but in much milder form. Both proximally and distally there was gradual transition from infarcted to normal areas. On opening, the mucosa was hyperemic and the lumen was filled with dark bloody fluid. No ulcers were seen. The mesentery was not involved, but its lymph nodes were swollen.

The sigmoid colon, which was not involved in the infarction, showed diverticulosis and diverticulitis. Other findings included postoperative pelvic adhesions, early portal cirrhosis, cortical adenoma of left adrenal, very slight arteriolar nephrosclerosis, and nodular hyperplasia of thyroid. The lungs showed only slight right lower lobe congestion. The heart (weight 330 grams) was normal except for a few small subendocardial petechiae. The brain was normal. The aorta, cerebral, coronary and all other arteries showed little or no arteriosclerosis.

Microscopic Findings Sections of the superior mesenteric vessels showed normal vein and slight intimal thickening of the artery. Sections of the infarcted intestines showed hemorrhages and severe vascular congestion (most intense in submucosa) (Figures 2 and 3). The mucosa showed scattered foci of superficial erosion and early necrosis. There were only a few scattered inflammatory cells in the intestinal wall. Numerous sections failed to reveal thrombosis or vascular disease.

Discussion The autopsy revealed no clue to the etiology of the massive infarction of most of the small intestine and part of the large intestine. The infarcted areas were clearly in the distribution of the superior mesenteric artery yet this artery and its accompanying vein were found patent. Nor was there cardiac or vascular pathology that might have predisposed to thrombosis or embolism. The extent and distribution of the infarct ruled out the possibility of intussusception[20] or volvulus[21] with spontaneous reduction before death.

The possibility of arterial spasm must be considered, especially in view of the associated hypertension, just as spasms must be considered when myocardial and cerebral infarcts are found without accompanying vascular occlusion. Berman and Russo[22] considered mesenteric vascular spasm as the mechanism of "abdominal angina."

However, experimental studies have shown other mechanisms for producing infarcts without vascular occlusion. Grégoire and Couvelaire[8] have produced intestinal infarcts in dogs by an immunological mechanism (Arthus phenomenon). Dogs were subjected to multiple injections of an antigen (horse serum) directly into the intestinal wall and these injections were repeated at eight day intervals. The first two operations had no effect, but after the third procedure the injected loop of intestine became discolored purple or even black. The arterial pulsations ceased and the veins became distended by dark blood. There were also systemic manifestations attributed to anaphylaxis such as dilation of pupils and secretion of abundant mucoid saliva.

We have described an immunologic method for producing intestinal infarcts in guinea pigs.[23] We took advantage of the fact that guinea pig organs contain the Forssman antigen. This antigen is called "heterophile" because it occurs in the tissues of many different species, for example, in the horse, dog, cat, mouse, guinea pig, chicken, tortoise, and some fish. It occurs in the erythrocytes (but not in the tissues) of sheep and goat, and it also occurs in certain bacteria. The rabbit does not contain Forssman antigen, therefore it reacts to the injection of Forssman antigen by formation of antibodies. We produced Forssman antiserums by injecting either guinea pig kidney extract or sheep erythrocytes into rabbits. The Forssman antiserums proved toxic if injected into animals like the guinea pig which contain Forssman antigen. Thus intradermal injection caused local necrosis[24] while intravenous injection caused a type of anaphylactic reaction (termed "reverse anaphylaxis") probably due to toxic effect on the lungs.[24] Forssman himself practised intra-arterial injections and produced brain damage in guinea pigs by carotid artery injections.[25] Graña produced pancreatic necrosis by injection into the pancreatico-duodenal artery of dogs.[26]

We injected Forssman antiserums into the small intestinal arteries of guinea pigs, choosing the last branches before the terminal arcade. The injected artery and its accompanying vein were then ligated, but the terminal arcades provided ample collateral circulation, as proved by many control experiments. During the injection a portion of intestine blanched due to the antiserum mechanically washing out the blood, but this temporary blanching had no harmful effects as proved by control injections of saline or normal serum.

The animals were killed after twenty-four hours and hemorrhagic infarcts were uniformly found. The distribution of the infarcts corresponded to the distribution of the arteries into which Forssman antiserum was injected. The infarcted segments were swollen and dark reddish-purple, and the lumens contained bloody fluid. Microscopic examination showed necrosis, ulceration, hemorrhage and congestion. No arterial thrombi were seen, and only occasionally was a venous thrombus detected. The Forssman antiserums caused infarcts even after considerable dilution (up to 1 to 80 for the strongest serum), but after further dilution only focal ulceration and congestion were produced, and still more dilution abolished the reaction completely.

We proved that the infarcts were caused by a specific antigen-antibody reaction by showing that an antiserum whose Forssman antibodies were removed by a test-tube absorption procedure lost its ability to produce infarcts. Furthermore, normal serum and several unrelated immune serums failed to produce infarcts. We also noted that a Forssman antiserum which produced infarcts in the guinea pig was also effective in the cat, due to the occurrence of Forssman antigen in these two species, while it was ineffective in the rabbit. Finally, we noted that the relative potency of several Forssman antiserums in producing infarcts was roughly parallel to their test-tube titers against sheep erythrocytes.

By what mechanism does this immunologic reaction cause an infarct? The Forssman antigen occurs in the blood vessels including the endothel-

ium.[27] The union of antibody and antigen might result in endothelial damage with increased permeability leading to edema, stasis, diapedesis of red cells, and finally hemorrhage and necrosis.

However several workers who have studied intestinal infarcts produced by the Arthus phenomenon have invoked a neurogenic mechanism. Longo et al[28-31] have reported that extirpation of the vegetative nervous system or intravenous procaine prevents experimental infarcts of Arthus type. These workers believe that the immunologic reaction leads to capillary dilation and infarction by way of axon reflexes in autonomic nerve fibers. In this connection, it is pertinent to point out that Reilly et al [32] have produced intestinal infarcts by injections of various toxins and chemicals in the vicinity of the splanchnic nerve. Indeed, Gregoire and Couvelaire[8] believe that irritation of the autonomic nervous system is the common denominator linking their immunologic experiments with the work of Reilly et al.[32]

It is not certain whether the neurogenic mechanism applies to our immunologic experiments with Forssman antiserums, and it is still less certain whether these considerations apply to intestinal infarcts in humans. However, the experimental evidence combined with clinical and autopsy observations indicate that intestinal infarcts can and do occur in the absence of vascular occlusion. This conclusion appears to have practical applications as there are numerous reports of intestinal infarcts discovered at laparotomy which responded favorably to intramuscular adrenaline (1 mg.)[4,6,7,8,9,13,14,16] or to intravenous procaine (1%, 15-20 cc)[6,10,11,28,29,31] or even to general anesthesia alone.[8]

Summary

The autopsy on a case of intestinal infarction without vascular occlusion was described. Although the etiology and pathogenesis of this condition are not known, it can be reproduced in animals by several methods, and it may be amenable to therapy with intramuscular adrenaline or intravenous procaine.

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RESEARCH GRANT

The International Academy of Proctology announces a $1,000 research grant to Dr. Seymour Levine for a continuation of his present research project relating to ulcerative colitis. A review of this project will be made at the Twelfth Annual Teaching Seminar, April 23rd through April 28, 1960, the Americana Hotel, Miami Beach, Florida.

Pneumatosis Cystoides Intestinorum Hominis

Part II

The title of this presentation "Pneumatosis Cystoides Intestinorum Hominis" will be interchanged often with the following terms: pneumatosis, pneumatosis intestinalis, intestinal pneumatosis, blebs, blisters, vesicles, gas cysts, intestinal gas cysts, intestinal blebs, intestinal vesicles.

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Review of Ancient Medical History It took centuries to overcome the theories and doctrines of the ancient Grecians, Hippocrates, Asclepiades (124 B.C.) and Galen (131-201 C.E.) which "effectively prevented any scientific investigation of pathological anatomy."

"The Greek school, as well as other ancient and medieval schools, had no conception of pathological changes of tissues in a diseased body. They did not suspect that structural changes might appear in a patient's organism."

Scientific investigation, based upon tissue pathology and anatomic changes was prevented for a period of fourteen centuries, due to the acceptance of Galen's doctrine on diseases.

In referring to ancient medical history, it is disclosed that the ancient Hebrews of the Talmudic era of the Land of Canaan and Babylonia, can be credited with originating the science of pathology, 2,000 years ago, *By Their Discovery That Diseases May Be Associated With Morphological Changes in the Organs of the Body and Become Manifest by Structural Alterations and Functional Abnormalities.*

Structural alterations were the basis of their doctrine; in the main, it conforms with the theories promulgated by modern science.

The anatomic structural and histologic changes of the organs resulting from diseases were the fundamentals of the Talmudic doctrine. Their great knowledge of anatomic pathology was mainly obtained from the observation of slaughtered animals, on the basis of color, position, consistency, growth and the presence of lesions. They were able

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to ascertain the course and lethal determination of many diseases on the basis of the nature of the pathological condition." The main reasons for this great historical oblivion of medical progress for a period of twenty centuries was due to the fact that the then existing physicians could not read the Talmud and were not able to understand the Babylonian teachings of that particular time. The Talmud was rather considered as a "Corpus Juris of ritual law,: rather than "a treatise on medicine."

The history of medicine and the progresive knowledge obtained in animal pathology (which may even be referred to Pneumatosis Cystoides Intestinorum Hominis and Animalis,) reveals that Rudolph Virchow and Julius Cohnheim, nineteen centuries later, finally rediscovered pathologic anatomy as the basis of our modern teachings.

The great acquaintance of the ancient sages of Babylonia with visceral pathology, obtained from inummerable autopsies made on domestic animals, contributed enormously to the progress of comparative anatomy, experimental surgery and diagnosis.

According to Garrison the ritual "autopsies made by the ancient Hebrews upon slaughtered animals to distinguish the fit for food from the unfit for food, threw a light upon pathological appearances which the ancient Greeks never gained."

Due to these ritual laws of the ancient Hebrews the Sages of the Talmud studied attentively the pathology of the abdominal and thoracic organs.

It would be of great interest to review the special treatise named "Hulin" compiled by the ancient Hebrews, describing numerous pathological entities found in multiple autopsies performed upon different animals. Such investigation could possibly reveal the presence of Pneumatosis Cystoides Intestinalis Animalis as long as twenty centuries ago. If this is the case, the existence of Pneumatosis is probably as old as the creation and formation of the intestinal tract.

Review of the Literature The first described case of Pneumatosis Cystoides Intestinorum as encountered in vivo during a laparotomy was reported by Hahn in 1899. Finney described the first case of pneumatosis in the United States in 1908. The earliest attempt to analyze the content of gas cysts was made by Mayers in 1825, who established the presence of carbon dioxide, nitrogen, and oxygen; however, up to the present time great discrepancies as to the percentage of the components persist. According to most investigators the variance of percentage in the gaseous substance of the blisters is attributed to the duration of exposure to the environment before analysis was undertaken.

In 1913, the very first x-ray diagnosis of pneumatosis was made by Barjon and Dupasquier and the earliest x-ray interpretation in the United States was made by Lerner and Gazin in 1946.

During the discussion of a paper on pneumatosis presented by J. Edwin Alford in the Buffalo General Hospital, Buffalo, New York in 1955, Raymond J. Jackman in reviewing this disease made a number of interesting remarks. According to Jackman two hundred and sixty cases of pneumatosis have been reported in man, however less than fifty cases were confirmed to be authentic. In continuing his discussion about this disease he added that "the fact that we saw two patients with this relatively rare

and interesting entity within a short time after it was brought to our attention by Dr. Alford's paper, makes me wonder whether the condition is not more common than is generally believed."

The reported cases by Jackman and Alford are of particular interest to the proctologist, as the main complaint and outstanding symptoms of their reported cases, was rectal bleeding.

Often patients with similar symptoms are seen in the office complaining of intermittent bleeding and pain in the lower bowel; they are usually examined and if hemorrhoids are found and bleeding persists for some time, a hemorrhoidectomy is usually performed. Rectal bleeding often accompanies a number of pathological entities located in the anus, rectum, sigmoid and descending colon; ergo, it is evident that diagnosis is of vital importance. As time progresses our diagnostic armamentarium improves, facilitating and enabling us to establish the correct diagnosis.

A. W. Marino, while discussing Alford's paper was of the opinion that "The clinical diagnosis of Pneumatosis Cystoides Intestinalis is quite difficult. Of the forty-five cases already in the American literature only five were diagnosed preoperatively . . . ."

When referring to Alford's presentation, Walter Birnbaum asserts that recent studies made in animal pathology may have some bearing on pneumatosis cystoides hominis; he further affirms a number of recent papers dealing with Histoplasmosis in animals, and particularly in swine, disclose that such affected animals present submucosal and subserosal gas cysts, as frequently encountered in the human kind of pneumastosis cystoides intestinorum. There is sufficient evidence that animal Histoplasmosis is easily transmitted to man.

Further interesting remarks were made by Ernest N. C. McAmmond of Vancouver, B. C. on a paper, describing two of his cases presented in Geneva, in 1955 and subsequently published the same year in a Canadian medical journal. One of his cases was diagnosed at the operation, and the second case preoperatively showing typical radiologic evidence of gas cysts.

According to McAmmond there are three typical findings which contribute to the diagnosis of Pneumatosis. In citing them he claims that in one patient the affection was located in the R.L.Q. and in the second case it was found to be present in the left side of the colon. The radiologic diagnosis of gas cysts in the left side of the bowel was subsequently confirmed by sigmoidoscopy. It is interesting to note that his cases also presented symptoms usually diagnosed as proctitis, sigmoiditis, diverticulitis, or ulcerative colitis. The most important features of the second case consisted of lower abdominal pain accompanied by the passage of blood and mucus. During the sigmoidoscopy thirty to forty cysts were pricked with a high-frequency needle and all of them exploded with a loud puff. A similar experience was obtained by Jackman when referring to his case report.

The experience of many observers confirms that intestinal pneumatosis is a benign disease. Often there is disappearance of symptoms with apparent complete recovery. However, it was observed that recurrences often appear after a year or more of complete freedom from symptoms. The significance of periodic flare-ups is not easy to comprehend.

In the field of dermatology there are a number of particular allergic eruptions which appear periodically in the form of bullae or vesicles on the skin, mucosal surfaces of the mouth, linings of the nose, and anal outlet.

Arthur E. Lewis, of Seattle, Washington, reports a very unusual case of a 32 year-old female with a history of constipation which became much more accentuated following a prefrontal lobotomy. Examination revealed fecal impaction, and adequate preparation with castor oil and enemas was administered previous to the sigmoidoscopy. The endoscopic findings disclosed that the entire rectal and lower sigmoid mucosa was transformed in a mass of "bullae-like" formation of varying size from 1-½ to 3 cm. in diameter, with intraluminal protrusion. There was no evidence of bleeding and the involved rectosigmoid lining was pale and pinkish with no evidence of ulceration. The patient was rectosigmoidoscoped four days later and none of the previous bullae were found. The rectal and lower sigmoid mucosa were of normal aspect.

In discussing this unusual condition, Lewis was of the opinion that the chemical irritation of castor oil could have provoked an allergic edema of angioneurotic nature.

The proctologist who frequently encounters patients with rectal bleeding or other rectal affections may easily overlook the existence of pneumatosis either by not being aware of this disease or by attributing the bleeding to the concomitant presence of hemorrhoids. The importance of performing a routine proctosigmoidoscopy cannot be emphasized strongly enough. Such procedure would either establish or exclude the presence of rectal or sigmoidal pneumatosis, a diagnosis which otherwise might easily be missed.

To our knowledge there is little reference in any medical surgical or proctologic textbook referring to intestinal pneumatosis.

The so-called "rare diseases" may not be so "rare" at all; reference is made to pneumatosis and other uncommon maladies such as coccidioidomycosis which, until World War II, was recognized as a systemic disease affecting predominantly the respiratory system.

Etiology and Pathogenesis of Pneumatosis Cystoides Intestinorum Referring to the etiology of pneumatosis these seem to be differences of opinion as to its etiologic factor.

A great part of the published literature is dedicated to discussions and incertitudes of existing theoretical affirmations which could, or may, cause intestinal gas cysts. However, for the purpose of brevity, only the main features of these theories, Neoplastic, Nutritional, Bacterial, Chemical, and Mechanical will be cited.

NEOPLASTIC THEORY: The earliest theories attributing intestinal pneumatosis to neoplastic origin goes back to the end of the eighteenth and the beginning of the nineteenth century. This theory was based upon the belief of the existence of particular tumor cells within the intestinal wall, able to form gas cysts.

NUTRITIONAL THEORY: This theory is based upon the belief that pneumatosis is the result of chronic malnutrition.

Past and recent experiments on various animals reveals that a progressive, prolonged, administration of a starvation diet, reproduces intestinal gas cysts, as often observed in man. Such condition, however, reverts to normal as soon

as the diet is corrected.

BACTERIAL THEORY: The bacterial theory affirms that a number of intestinal gas-forming bacteria penetrate the intestinal wall, where gas cysts are formed. Some observers were able to obtain positive cultures; however, others did not succeed in isolating similar gas-forming bacteria.

CHEMICAL THEORY: This theory is based upon the fact that under normal physiological conditions the ingested or produced gases, during the process of normal digestion, such as oxygen, carbon dioxide and nitrogen, are usually absorbed by the intestinal mucosa and progressively combined with the various components of the blood stream.

Under certain systemic pathologic conditions these gases are not absorbed, remain stagnant, and are not assimilated from within the lymphatic channels. According to Masson and Severi the progressive accumulation of such gases become "blocked" in the lymphatic channels, causing the appearance of gas cysts. In fact, it is the latter author who suggested the term of "pneumocystic lymphopathy."

MECHANICAL THEORY: The mechanical theory is the most accepted one, and supported by a number of outstanding investigators. The formation of gas cysts, according to this theory, results from penetration of gas in the intestinal lumen through mucosal defects or existing ulcerations. To substantiate this affirmation it is shown that the majority of cases afflicted with pneumatosis occur in the presence of total or partial intestinal obstruction.

A New Theory: *Lymphatic Hypertension of the Gastrointestinal Tract and Circulatory Failure of the Lymphatic System.*

A lengthy description of the origin, role and distribution of the entire lymphatic apparatus in general and of the gastrointestinal tract in particular, is beyond the scope and limits of this presentation. However, the main physiological factors responsible for its normal flow and some of the mechanical interferences secondary to a number of organic affections, will be mentioned for the benefit of the reader before describing the above New Theory.

"THE CIRCULATION OF THE LYMPH: The direction of flow of the lymph is from the tissues toward the large lymphatic trunks, the thoracic and the right lymphatic duct. The flow is maintained in this direction mainly by a difference in pressure at the two ends. At the opening of the large trunks into the jugular veins the pressure is very low; in the vein, in fact, it may be zero or even negative as compared with the atmospheric pressure. The opening between the lymph vessel and the vein is protected by a valve which opens toward the vein, and the lymph, therefore, will flow into the vein as long as the pressure in the latter is lower than in the lymphatic duct.

At the other extremity of the system, in the tissue spaces to which the lymphatic capillaries are distributed, the pressure, on the contrary, is high. Its exact amount is not known, but, since the pressure in the blood capillaries is equal to 20 - 25 mm. Hg, the pressure in the liquid of the surrounding tissues must also be considerable. The tissues are, in fact, in a condition of turgidity owing to the pressure of the lymph in the tissue-spaces. This difference in pressure at the two ends of the lymphatic system is the main constant factor in moving the lymph.

The flow of lymph or chyle in the intestinal area is also, without doubt, greatly assisted by the peristaltic and especially by the rhythmic contractions of the musculature of the intestines, and by the independent movements of the villi. The volume of the lymph in this region is especially large and the lymph capillaries and veins are provided with valves. Rhythmical contractions of the musculature of the intestine must squeeze the lymph toward the thoracic duct, acting like a local pump to accelerate the flow of lymph. A similar influence is exerted by the contractions of the skeletal muscles.

After meals, during the period of absorption from the alimentary canal, the flow is much more abundant than in the fasting animal."

"Factors Controlling the Flow of Lymph We may adopt, provisionally at least, the so-called mechanical theory of the origin of lymph. Upon this theory the forces in activity are, first; the intracapillary pressure tending to filter the plasma through the endothelial cells composing the walls of the capillaries, and second; that form of molecular energy which gives rise to the phenomena of diffusion and osmotic pressure. By the action of this force, the flow of water from one place to another is influenced in accordance with the difference in concentration of the dissolved substances. These two forces acting everywhere control primarily the amount and composition of the lymph, but still another factor must be considered, for when we come to examine the flow of lymph in different parts of the body, striking differences are found. It has been shown, for instance, that in the limbs, under normal conditions, the flow is scanty, while from the liver and the intestinal area it is relatively abundant. In fact, the lymph of the thoracic duct may be considered as being derived largely from the latter two regions. Moreover, the lymph from the liver is characterized by a greater percentage of proteins. To account for these differences Starling suggests the plausible explanation of a variation in permeability in the capillary walls. The idea that the permeability of the capillaries may vary under different conditions has long been used in a pathology to explain the production of that excess of lymph, which gives rise to the condition of dropsy or edema. The theories and experiments made in connection with this pathological condition, have, in fact, a direct bearing upon the theories of lymph formation. Under normal conditions the lymph is drained off as it is formed, while under pathological conditions it may accumulate in the tissues owing either to an excessive formation of lymph, or to some diminution in the process of absorption, or to some interruption in its circulation.

For the foregoing considerations it is evident that changes in capillary pressure, however produced, may alter the flow of lymph from the blood-vessels to the tissues, by increasing or decreasing, as the case may be, the amount of filtration; changes in the composition of the blood, such as follow periods of digestion, will cause diffusion and osmotic streams, tending to equalize the composition of blood and lymph; and changes in the tissues themselves following upon physiological or pathological activity will also disturb the equilibrium of composition, and therefore, set up diffusion and osmotic currents. In this way a continual interchange is taking place by means of which the

nutrition of the tissues is effected, each according to its needs. The details of this interchange must of necessity be very complex when we consider the possibilities of local effects in different parts of the body. The total effects of general changes, such as may be produced experimentally, are simpler, and are apparently accounted for, so far as our present knowledge goes, by the physical and chemical factors enumerated."

Pathogenesis of Lymphatic Hypertension Metabolic and chronic intestinal diseases including pyloric stenosis and progressive intestinal obstruction usually cause severe forms of malnutrition.

Prolonged depletion of essential metabolites, electrolytes and lack of sufficient proteins, predisposes to infection, imbibition, edema and changes in the dynamics of the circulatory and lymphatic systems. Intestinal imbibition causes often enormous distensions of the abdomen, enlargements of the liver and spleen, hemopoietic disturbances and the formation of ascites. Such condition is usually present in chronic states of malnutrition and severe forms of avitaminosis; the poor population of the under-developed countries are frequently the victims of this clinical entity, the result of chronic starvation.

Many predisposing and existing factors, which accompany chronic intestinal diseases, are probably responsible for the presence of pneumatosis.

The progressive course of pneumatosis, often causes intestinal obstructions and paralytic ileus.

As the process progresses, all vital functions are gradually diminishing, including intestinal absorptions with all its deleterious effects on the lymphatic and circulatory systems. Pooling of intestinal lymph takes place and the engorgement of the channels continues, causing important hydrodynamic changes.

Another important factor contributing to lymphatic stagnation is the lessened contractions of the atrophied skeletal musculature which, under normal conditions, contributes to the onward flow of lymph to the major thoracic ducts.

Prolonged bed rest contributes noticeably to reduce peripheric drainage and further stagnation of lymph, thus increasing intraluminal pressure. This passive, progressive, increase of intraluminal pressure of the lymphatic channels causes valvular insufficiency and retrograde lymphatic flow, thus establishing diffusion and osmotic changes in the lymphatic system.

The shallow expansions of an emaciated chest, as seen in pneumatosis, reduces the necessary intrathoracic negative pressure needed to aspirate proper amounts of lymph in order to facilitate its flow and prevent the increase of abdominal endolymphatic pressure.

The progressively augmenting abdominal endolymphatic pressure finally assumes hypertensive proportions with increasing valvular insufficiency. Such a condition is often observed in pulmonary hypertension. The increasing lymphatic hypertension ultimately degenerates into congestive failure with turgidity of vessels and formation of edema and ascites.

The simultaneous presence of the described pathological triad in pneumatosis intestinorum, consisting of ascites, intestinal gas cysts, and the oily foam-like texture of the transudate, may be easily explained if we consider the described

pathogenesis of the progressively increasing endolymphatic pressure as the main causative mechanical hydrodynamic factor of the formation of the chyloperitoneum like ascites, edema, cystic formation of the intestinal wall and the presence of pneumoperitoneum.

A similar sequence as already briefly mentioned, takes place in a progressively increasingly pulmonary hypertension, where transudate and edema successively augment, creating congestive failure, thus becoming the predominant clinical picture.

Transformation of Intestinal Lymphatic Segments into Gaseous Intestinal Blebs A great number of investigators including the author are of the opinion that the intestinal lymphatic channels are the seat of gas formation in Pneumatosis Cystoides Intestinorum Hominis.

The exaggerated production of carbon dioxide, resulting from excessive fermentation of residual aliments and chyme, stagnant in the bowel and exposed to the mercy of the intestinal flora, may explain the mechanism of gas cyst formation in the lymphatics.

The two main gases present in a distended bowel are carbon dioxide and oxygen; however, their great differences in volume and solubility make it evident why the gaseous content of the cyst is mostly carbon dioxide. The following chemical intrinsic properties of these two gases reveal that: *CO_2 is about 25 times as soluble as O_2, and its pressure gradient is 1/10th that of O_2.*

Anatomic Peculiarities of Lymphatic Channels Lymphatic vessels present an anatomic peculiarity similar to the arrangement of buddings observed in the reproductive elements of the lower organisms, as seen in protozoan or cryptogamic plants. (See Fig. 18. page 197, June 1959 issue.)

A number of investigators in the past were of the opinion that the walls of the gas cysts are dilated lymphatic structures. Masson made the suggestion that "in the presence of intestinal fermentation, carbon dioxide is absorbed in the lymphatic channels and dissolved in the lymph to a point of saturation. Because of the slow circulation of the lymph the gas is blocked in the lymphatics and forms cysts." Severi agrees with Masson on the important role the channels play in the absorption and diffusion of gases. He suggests the term "pneumocystic lymphopathy."

The chemical theory based upon the absorption of gases as the etiologic factor of Pneumatosis Intestinalis is the most acceptable and seems to explain satisfactorily the process of gas blocking and bleb formation. However, the pathogenesis of Pneumatosis Intestinalis apart from the underlying factors of excessive gas production in the intestine and consequent lymphatic absorption, the formation of gas cysts, pneumoperitoneum, and transudate are probably also the result of a condition which may be termed "lymphatic hypertension" as previously mentioned.

Microscopic examination of an intestinal gas cyst discloses that its walls are lined with flattened epithelium and that such structure resembles a dilated segment of a lymphatic capillary.

The term "pneumocystic lymphopathy" as suggested by Severi is a leading thought which paves the way to the author's new theory of "Intestinal Lymphatic Hypertension" as described in this paper.

Treatment To suggest or advise adequate treatment for such a complex

pathological entity and variant syndrome, is not only practically impossible, but medically improper.

Regardless of the established clinical symptoms and pathogenesis of the involved bowel, the etiologic factors are often multiple and of unknown origin.

The only therapy which can be attempted is the correction of altered metabolic processes and the remedying of the malnutritional status.

Great attention is to be given to the patient's personality changes, due to the fact that they do not follow medical advice regardless of their good intentions and willingness. This behavior may be attributed to a type of lacunar amnesia, which seems to be a part of the entire syndrome.

Due to the frequent occurrence of intestinal obstruction, surgical correction is to be performed, without hesitation, as soon as the diagnosis is established. Complete exploration of the entire gastrointestinal tract is mandatory as multiple occlusions are frequently present.

Special attention is to be directed to the afferent ansa of the jejunum and its mesenteric attachment just under the ligament of Treitz and around its entire semilumar curve. In this area strangulation of the mesentery is frequently observed.

Comments and Conclusions The described case report reveals that this patient was submitted to surgery in 1953 with a diagnosis of "Cholecystoduodenal fistula with impacted gallstones in the ileocecal valve and with intermittent intestinal obstruction." The postoperative diagnosis was "non-functioning gallbladder, generalized peritonitis, etiology undetermined."

The gastrointestinal series, preoperatively, show pronounced pneumoperitoneum. Seemingly the existing pathology of pneumatosis was not recognized. Postoperative scout films, taken ten to fifteen days later, confirmed marked diminution of the amount of pneumoperitoneum, but a small amount of air was still visible under the right diaphragm. The decrease of air was probably due to the presence of the two catheter drains inserted in the R. U. and R.L.Q. as their presence was noted by the radiologist.

February, 1957, the diagnosis of "serous cystic disease of the mesentery" was made by the radiologist, for the first time. An intestinal obstruction developed in March 1957, and the operative findings were "generalized pneumatosis of the small bowel with the formation of large granulomas consisting of cluster-like formations of innumerable gas cysts." It was found that one of these granuloma-like formations, situated in the terminal ileum, about fifteen to twenty inches above the ileocecal valve, was intussuscepted. Disengagement was not possible and segmental resection was performed with an uneventful recovery.

It is noteworthy to mention that on the third postoperative day subcutaneous emphysema was present in the incision and both inguinal regions. Scout films taken the tenth postoperative day revealed cessation of pneumatosis with no air under the diaphragms and non-visualization of gas cysts.

Three and a half months following the first operation a second laparotomy was performed due to a newly formed obstruction which was located in the R.U.Q.

Diagnosis of the second intestinal occlusion was made four weeks prior

to surgical consent. At the time of the operation patient's condition was critical. The operative findings were an intricated volvulus of the jejunum and found to be adhering to the right anterior hepatic region.

The patient expired fourteen hours postoperatively from a C.V.A. The autopsy disclosed strangulation of the afferent ansa of the jejunum with twisting of its mesenteric attachment. Seemingly, strangulation and twisting of the jejunum under the ligament of Treitz was overlooked. This area seems easily exposed to edema and vascular interference due to the small outlet around the ligament of Treitz for the jejunal passage and free accommodation of its originating ansa.

The stomach was found to contain a pronounced stenosis of an hourglass formation in the antral region, as evidenced in the illustrations. The gastric stenosis was not reported in the 1953, nor in the March 1957 surgical procedures. During the March 1957 operation, regardless that the stomach was examined, no stenosis was noted. The omission was probably due to the presence of dense adhesions of the stenotic area. Repeated gastrointestinal studies, made preoperatively, did not reveal the presence of gastric stenosis, and consequently the dense adhesions on the antral region were not disturbed.

The main obstruction found at the time of the operation was located in the terminal ileum.

The patient's general condition was such that a short procedure was advisable.

It is definitely evident that regardless of locating an existing obstruction, a complete and thorough exploration of the entire gastrointestinal tract is to be made, even if patient's condition is not too promising.

Close observations made for a period of three and a half months definitely established that inanition is one of the predisposing factors of pneumatosis. Furthermore, it can be concluded that after installing the proper nutritional regime, malnutrition can be controlled and gradual restorative improvement can be expected.

The fact remains that cessation of pneumatosis can take place spontaneously and in our case, it followed a segmental resection of the intussuscepted ileum without disturbing an existing gastric stenosis, which was found accidentally postmortem.

Referring to the etiology of pneumatosis intestinalis and the prevailing theories, it seems that a combination of the mechanical, nutritional, and chemical theories are causative factors of the pathologic entity.

The formation of gas cysts is probably the result of a disturbed lymphatic circulation which takes place in the entire gastrointestinal tract.

The distended gaseous cysts resemble, morphologically, lymphatic formations, and their contents are probably derived and refilled from the excessive formation of carbon dioxide.

The metabolic and electrolytic changes of the circulatory system are possibly responsible for the osmotic changes that occur in the gastrointestinal tract.

Pressure changes in the lymphatic net of the gastrointestinal tract in general, and the intestinal wall in particular, cause lymphatic stagnation with edema, and the formation of abdominal transudate resembling intestinal chyme.

This condition is probably the result

of congestive lymphatic failure, established by progressively increasing endolymphatic pressure, assuming, finally, hypertensive proportions, and can be compared to congestive heart failure resulting from a steadily increasing pulmonary hypertension.

The most common symptoms of diagnostic importance in intestinal pneumatosis are chronic pneumoperitoneum, frequently reported by other observers and presented herein, for a period of four years. The presented case revealing chronic pneumoperitoneum for a number of years may have been present for some time before it was ever detected. It is evident from this and other patients' histories that free air in the abdomen is a very early diagnostic symptom long before other clinical subjective phenomena appear. It is reasonable therefore that great efforts be made to look for the presence of minimal pneumoperitoneum when vague disturbances of the gastrointestinal tract appear. Pneumoperitoneum without the presence of shock air and the alarming symptoms of a perforated hollow viscus, shall always remind us of pneumatosis. X-rays, showing "honeycombed" configurations of abdominal viscera shall suspect the presence of pneumatosis. Roentgen evidence of the disappearance of pneumoperitoneum and gas cysts was confirmed by a second laparotomy; and is usually a very reliable examination of which greater diagnostic use is to be made.

The case presented in this paper corroborates that the disease is of chronic nature and may continue for years. Pneumatosis is usually considered a benign affection, however, the often befalling intestinal obstruction makes its prognostic outlook not too encouraging. The site of the jejunal strangulation with its twisted mesentery was found to be free of gas cysts during the March 1957 exploratory. There is a possibility, however, that gas cysts may have been present in the posterior aspect of the ligament of Treitz or at the duodenojejunal junction.

Where it would be technically impossible to detect, due to the peculiar anatomic formation, the function of the visceral and parietal serosa forming ligaments, the presence of such gas cysts may cause circulatory interference by way of pressure to the vascular pedicle of the jejunal-mesentery and consequently to the jejunum itself, causing overdistension and volvulus.

The first apparent March 1957 obstruction was caused by intussusception of a granuloma-like cluster formation of overlapping gas cysts in the distally situated dilated gut. The topographic location of most gas cysts were found on the antimesenteric surface thus corresponding to the typical anatomical distribution of the lymphatic aggregates.

The invariably steady findings of epithelial cells, lining the walls of the gas cysts and the topographic multiplicity of the blebs, definitely resemble the anatomic formations of the lymphatic segments.

The presence of ascitic fluid of foamy and glycerine-like consistency encountered in the described case was also observed by other investigators, however, the factors of consistency and foaminess were not mentioned.

Summary

1. A review of the world literature of Pneumatosis Cystoides Intestinorum Hominis, with a case report, is presented. It occurs rarely in man and is of unknown pathogenesis.

2. Formation of gaseous cysts on the intestinal visceral serosa and through the intraluminal mucosa occurs along the gastrointestinal tract as a result of this pathologic condition. A foamy, oily transudate is a concomitant finding.

3. The disease is chronic, of unusually long duration, and progresses slowly, causing emaciation, invalidism and personality changes. Presenting symptoms are those of chronic progressive intestinal obstruction.

4. Etiological theories are explored. A working hypothesis of lymphatic hypertension is formulated.

5. Segmental resection of intussuscepted terminal ileum resulted in arrest of cyst formation and general improvement of patient.

6. Intervention for strangulated jejunum, three months after resection, was delayed, by patient, beyond safety.

7. Autopsy showed good results of first laparotomy and revealed jejunal strangulation. A presumptive diagnosis of a cerebrovascular accident, before or during surgery, was made.

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Resumen

1. Se presenta una revisión de la literatura mundial sobre Pneumatosis Intestinorum Hominis, con un caso clinico. Se presenta rara vez en el hombre y es de origen desconocido.

2. Como resultado de esta condición patológica se forman quistes de gas en la serosa visceral del intestino

y a traves de la mucosa. Se encuentra ademas un exudado aceitoso espumoso.

3. El padecimiento es cronico, de duración exageradamente larga, progresando lentamente, cuasando emaciación, invalidez y cambios en la personalidad. Los síntomas son de obstrucción intestinal crónica progresiva.

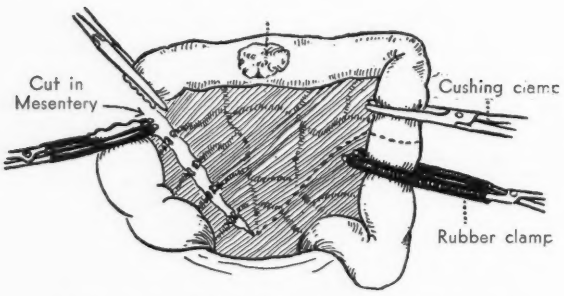
4. Se exploran las teorías etiológicas. Se formula una hipótesis sobre hipertensión linfática.

5. La resección segmental del ileum terminal invaginado, resultó util para contrarrestar la formación de quistes y para mejorar el estado general del paciente.

6. Tuvo que operarse en forma retardada tres meses mas tarde, por cause del enfermo, de un cuadro de estrangulación yeyunal, mas allá de los limites de seguridad.

7. La autopsia mostró buenos resultados de la primera operación y reveló la estrangulación del yeyuno. Se hizo un diagnóstico de presunción de accidente cerebro-vascular, anterior o simultaneo al acto quirurgico.

Clini-Clipping



Method of excising involved portion of bowel before making an intestinal anastomosis.

The Use of an Intravenous Fat Emulsion

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Parenteral nutrition using intravenous fat emulsions has been under investigation for many years.[1, 2] Since World War II we have witnessed the advent of far reaching advances in parenteral fluid therapy. A wide variety of carbohydrates, proteins, and amino acids are generally available. By simple mathematical calculation we are able to improvise electrolyte solutions to cope with almost any situation that may arise. One of the presently existent needs in intravenous fluid therapy is an efficient source of calories to help prevent breakdown of tissue protein. It has been shown that seriously burned patients need at least four thousand calories daily if they are expected to heal well. Using the presently available parenteral caloric sources to meet this requirement, ten liters of a ten percent carbohydrate solution would have to be administered. This we all know is impractical from several standpoints.

The majority of our seriously ill patients on fluid therapy are usually in negative nitrogen balance.[3] How then, can the most seriously ill be expected to heal well? Those patients with malabsorption syndromes, those patients with extensive burns, those patients with short-circuiting abdominal procedures are among a small, but select group who would benefit from a potent parenteral caloric source.

Stare and his co-workers[4-10] have been studying the problem of parenteral fat nutrition for many years. Their work indicates that lipid emulsions can be successfully prepared and infused with a high degree of safety, and above all, can be effectively metabolized. Mueller[11, 12] recently has extended these observations.

In our study we have approached the problem from another standpoint. We have been impressed that the patients who would need such therapy are among those most seriously ill. Our observations were made to determine the clinical efficacy of a commercially prepared lipid emulsion that could be administered in the same fashion as any other form of fluid therapy. The preparation employed in this study was Lipomul-IV* (11612 Series). Table I shows the composition and caloric potential of this product. It will be noted that each six hundred milliliter unit is capable of producing nine hundred and six calories.

The emulsion was administered to a

* Kindly supplied by Dr. E. A. Hawk, The Upjohn Co., Kalamazoo, Michigan.

TABLE I

CONSTITUENTS AND CALORIC VALUE OF LIPOMUL IV

	per 100 cc. Grams	per 100 cc. Calories	per 600 cc. bottle Grams	per 600 cc. bottle Calories
Cottonseed Oil	15	135	90	810
Glucose	4	16	24	96
Soyabean Phosphatide	1.2	—	7.2	—
Pluronic F 68	0.3	0	1.8	0
Total		151		906

TABLE II

SUMMARY OF INTRAVENOUS FAT EMULSION INFUSIONS

Patient	Diagnosis	No. of Infustions
M.S	Ulcerative Colitis	2
N.L.	Bronchogenic Carcinoma	13
J.W.	Gastric Carcinoma	10
O.N.	Malnutrition	7
L.S.	Gastric Carcinoma	26
M.Y.	Colonic Carcinoma	1
E.B.	Metastatic Ca., site?	20
M.G.	Rheumatoid Arthritis	2
M.R.	Pancreatic Carcinoma	1
M.P.	Tertiary Lues, Cirrhosis	1
A.M.	Possible Addison's Disease	1
B.	Gastric Carcinoma	4
J.	Bronchogenic Carcinoma	3
A.K.	Bronchogenic Carcinoma	6
R.S.	Osteogenic Sarcoma	3
B.S.	Pancreatic Carcinoma	7
R.S.	Ovarian Carcinoma	7
D.C.	Carcinoma of Breast	1
Total 18		115

TABLE III

REACTIONS TO IV FAT INFUSIONS

Reaction	Frequency
Unpleasant taste, base of tongue	1
Flush	5
Pain, Injection Site	1

group of patients with a variety of debilitating diseases as shown in Table II. A total of one hundred and fifteen infusions were administered to this group. One patient received a maximum of twenty-six daily infusions. Table III shows the variety of reactions to this type of therapy as noted in our series. The routinely recorded temperature, pulse, respiratory rate, and blood pressure were notably unaffected in our patients. It is interesting to note here, that others have reported alterations in the vital signs following intravenous fat infusions. The latter experiments were designed however, to determine what effects the infusions would produce in patients, and as a consequence they were observed much more rigidly for such changes. Our study was designed to utilize the routine hospital procedures and observations made on all patients receiving fluid therapy.

In our series seven patients expired during the course of the hospitalization in which they received the lipid infusions. Post-mortem examination in four failed to reveal any deleterious effects attributable to the fat.

The prime factor that will determine the eventual place of parenteral fat therapy will be the availability of the material to the metabolic mill. The work of Stare, Meuller, Levine,[13] and Upjohn[3] on the protein-sparing effects of fat emulsions is quite conclusive. We have had the opportunity to observe a mildly diabetic male who underwent laparotomy for obstructive jaundice due to carcinoma of the head of the pancreas. During the immediate post-operative period his diabetes was easily

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Presented at the Tenth Annual Teaching Seminar in Mexico, April 1958.

controlled with small insulin supplements to his parenteral fluids. We decided to give him some intravenous fat since we felt it would hasten his recovery, as this patient had lost nearly a third of his total body weight prior to surgery. He was given a daily infusion of one unit of fat emulsion. Almost immediately he demonstrated glycosuria, and hyperglycemia necessitating additional insulin coverage. This simple but clearcut observation is indicative of efficient utilization of this caloric source.

Basic physiologic studies with the material used in this study by Meyer,[14] et al., reveals that when administered properly no untoward cardiovascular or respiratory effects are observed. Hematologic and hepatic functions also were not markedly altered in chronic animal experiments.

The fat is cleared from the blood quite rapidly, usually within six to eight hours. We have noted a concomitant fall in serum cholesterol levels, with a rapid return to pre-infusion values.[15]

Human toxicity has not proven to be a serious problem with this preparation. It is important to stress that the presently available data suggests that the material should only be used when a serious caloric deficit exists and that treatment with fat infusions should be limited to short periods. There have been reports[16, 17] of an overload syndrome in patients who were given more than twice daily infusions for several weeks consecutively. This syndrome is characterized by fever, anorexia, sore throat, right upper quadrant pain, hepatosplenomegaly, jaundice, anemia, thrombocytopenia, bleeding, abnormal liver function tests, and sudanophilic material in the liver sinusoids noted on liver biopsy. This syndrome probably represents an over load to the normal fat pathways with resultant reticulo-endothelial phagocytosis of the excessive fat particles as foreign bodies. The solution to this problem at the present time is to limit the number and duration of infusions.

Annoying but not serious side-effects to the effusions are usually pain, dyspnea, flushing, or urticaria. These can frequently be obviated by starting the infusion slowly and gradually accelerating the rate of administration.

Discussion We are all aware of the early onset of malnutrition in our seriously ill patients. This problem is not particular to bowel-disease alone. However, some of the more serious nutritional deficiencies exist in diseases of the colon. Sleisenger[18] has observed that patients with ulcerative colitis or regional enteritis rapidly become malnourished and demonstrate an abnormal fat-protein loss. Patients with severe diarrheal disease of any etiologic cause, in whom oral alimentation enhances the diarrhea, and patients who lose large quantities of nutrients due to vomiting or intubation all become rapidly malnourished and begin to destroy tissue protein. Patients with artificial stomata, who lose large quantities of nutrients in the post-operative period also go into negative nitrogen balance easily. Patients with bowel malignancy are frequently temporarily inoperable due to severe wasting. In addition, patients with cardiovascular, respiratory or renal disease could be managed more easily in situations necessitating fluid therapy. This group and numerous others all are candidates for improved parenteral nutrition providing more calories than presently available.

Summary

1. A stable, utilizable intravenous fat source has been developed.

2. Administered with reasonable caution this agent appears to be safe.

3. Fluid therapy can now supply sufficient calories to prevent severe nitrogen depletion.

4. Systemic reactions in our series were negligible.

5. For the present this material should be restricted to therapy of no longer than two-weeks duration.

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Resumen

1. Se ha preparado una solucion estable de grasa para uso endovenoso.

2. Administrada con cuidado razonable, parece ser un agente seguro.

3. La terapia liquida puede en la actualidad proporcionar suficientes calorias que eviten una severa deplecion nitrogenada.

4. Las reacciones generales en la serie de estudio fueron minimas.

5. De acuerdo con los conocimientos actuales este medicamento debe usarse durante periodos no mayores de dos semanas.

The Use of Phenylbutazone and Feramid® in Acute Thrombosed Hemorrhoids

Newer Developments in Proctology

DONALD C. COLLINS, M.D.
Los Angeles, California

Recently, it has been proposed to employ Phenylbutazone (Butazolidin®) and Feramid® in speeding up the often prolonged and painful convalescence of acute thrombosed hemorrhoids.*

Phenylbutazone, a pyrazolidine derivative, possesses marked anti-inflammatory and analgesic properties. It has no lytic properties and does not liquefy the blood clot. By its intercellular action, perivascular infiltration and edema are promptly reduced. External skin tag formation, commonly associated with thrombosed hemorrhoids, is minimized. Phlebitis is alleviated, consequent venous rupture is greatly reduced, and further thrombus formation is arrested. In the primary stage of this disease, it is recommended to perform an immediate exeresis of the recent thrombus. The subsequent prompt use of Phenylbutazone will largely prevent bothersome, common complications during convalescence.

Archambault and Archambault[1] very recently reported their conclusions on 415 patients treated with 600 mg. of Phenylbutazone, daily, by mouth, for the first three days; and then given a maintenance daily dosage of 300 mg. for the next six days. By using this drug for only nine days, specific drug toxicity is avoided. These authors were most enthusiastic over the absence of complications, the prompt regression of both edema and inflammation, the absence of new thromboses, the avoidance of vascular rupture, and the very remarkable degree of analgesia obtained with the use of this preparation during their nine day treatment schedule. Patients placed on this therapy were out of bed within several days, instead of weeks, as was their previous behavior with the generally used present modes of symptomatic supportative treatment.

Recently, a liver polypeptide, with a

* Butazolidin® (phenylbutazone) is 3,5-dioxo-1,2-diphenyl-4-n-butyl pyrazolidine. Marketed by: Geigy Pharmaceuticals, Ardsley, New York; red sugar-coated tablets of 100 mg. Feramid® marketed by: Heparamine Co. Ltd., 176 Juana Ave., San Leandro, California; in 5.0 c.c. ampoules.

molecular weight of about 11,000, capable of detoxifying guanidine to ammonia and urea *in vitro,* has been produced.[2] During the past several years this liver polypeptide has been tried experimentally upon humans. It is called Feramid. One of the surprising actions accidentally discovered while using this drug to treat arthritis, was its ability to relieve pruritus-ani and clear up anorectal inflammations. It is administered intravenously in 5.0 c.c. amounts, two or three times weekly, until the desired results have been obtained. Usually, the results are rather permanent.

It is hoped that these two new modes of therapy may be of value to proctologists in their daily practice, and to help them to better alleviate their patients' bothersome symptoms and shorten their convalescence.

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EDITORIAL

NUTRITION AND DISEASE

Every physician, and perhaps especially the proctologist, must give his patient the benefit of the newer knowledge in nutrition. Death does indeed enter through the mouth, and the ancient admonition to stop digging your grave with your teeth is even more applicable in today's world.

As a result of H-bomb explosions we are all inevitably doomed to an earlier death. The radioactive isotopes ingested with our food predictably will increase the number of deaths from bone cancer and leukemia during the next 28 years at least, and probably for a much longer time. The effect on general resistance to disease is not known, and this may prove to be even more destructive.

There is now some evidence as well that radioactive iodine released in such explosions may be an important element in the increased incidence of thyroid cancer in children and adults. Man has at last learned how to destroy himself, and inadvertently has already initiated his own mass-suicide.

Strontium-90, found in largest and most concentrated amounts in milk (as is radioactive iodine), acts like calcium in the body, and is ultimately concentrated in the bones. It is therefore imperative that milk be eliminated from the human diet at once. Research should be undertaken to find a method to remove such radioactive materials from milk so that this excellent food product may once again be returned to the normal diet. Until that is accomplished, however, it is urgent that all milk be eliminated from human diets.

The major element in milk that is not found in abundance in most other foods is calcium. Calcium may be supplied by mouth in liquid, syrup or tablet form for both children and adults. It is a very simple matter to calculate the amount of calcium in a quart of milk—the recommended daily supply for children—and the amount in a pint of milk—the recommended daily supply for adults. This is then easily translated into an appropriate calcium dosage for both child and adult. Just as vitamin supplements are used for the average child and for many adults, so mineral supplements should be added, and most especially calcium, phosphorus and vitamin D, to assure proper absorption and utilization of the calcium.

Much is being written on the subject

of obesity these days, and there is statistical evidence that the individual who is underweight is less prone to the development of malignancy than the overweight person. We may now safely say —in view of the radioactive isotopes in *all food*—that the less we eat the better our chance to escape cancer. This is obviously true for cancer of the bone and blood stream, and statistically true —as weight decreases—for all cancer. I can think of no better reason, at the present time, to place all our patients on a limited, reducing diet. It is therefore essential that proctologists, as well as all other specialists and general practioners, become aware of the importance of reducing the intake of all foods, as a prophylactic measure in the war against cancer. I am advocating that all physicians become nutritionists without delay. There is little point in skillful removal of hemorrhoids, fistulae, pilonidal cysts, etc., if your patients are then left to die from cancer of the bone, blood, etc.

And now we have the most powerful possible argument against over-eating—*all food is radioactive.* Every mouthful your patient eats is radioactive, and brings him a step closer to a cancer death. You need not worry about calories. Simply eat less and live!

I dwell at great length on this problem because the proctologist, in common with all other specialists, must not lose sight of the fact that he is primarily a physician. It is his duty to the patient to understand and treat him as a total person. If I may be permitted a bad pun, he should treat his patient as a whole and not as a hole. This is the psychosomatic approach to proctology, an approach that I have long advocated.

Nutrition is even more directly related to proctology in terms of the concept that colitis, celiac disease and sprue may be due to food sensitivity. Certainly it is true that proper control of diet is essential in the management of all such patients. The importance of diet therapy in the control of constipation and colitis need not be stressed to the proctologist and the gastroenterologist. And yet, I wonder how many prescriptions for laxatives, wetting agents, etc., are written daily by both proctologists and gastroenterologists, with almost total disregard for the basic importance of diet therapy.

We must not treat symptoms. We must recognize that "disease" is merely the patient's response to an irritating "substance" in his internal or external environment. We must treat the total patient in an environment—once again the psychosomatic approach.

In a previous article I have detailed my own approach to the stool study, as it relates to nutrition, a much neglected area in proctologic examination and diagnosis.

Many years ago at the Mayo Clinic, on an arbitrary basis, pilonidal cyst disease was added to the domain of the proctologist. I now advocate, *on a logical basis,* the extension of the field of proctology to include nutrition in all its phases. I advocate the psychosomatic, non-elementalistic concept of proctology rather than the presently archaic surgical approach. I advocate that the proctologist once again become a complete physician treating a total patient.

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Physical limitations of space in journal advertising make the presentation of all relevant data impractical, therefore, it is suggested that for suitable background on dosage indications and contraindications the standard package insert or more extensive background data be consulted.

The acceptance of material for advertising is based upon several criteria; for example, in respect to safety, all new drugs are required to correspond with the accepted Food and Drug application.

It is suggested that any differences of opinion of individual physicians with any advertisements be called to the attention of the editor, with a duplicate copy of the letter to the pharmaceutical house whose advertisement is the subject of the letter.

THE PUBLISHERS

BOOK REVIEWS

"SURGERY OF THE COLON" by E. S. R. Hughes, M.D., M.S., (Melb.), F.R.C.S., (Eng.), F.R.A.C.S., Hon. Surgeon, The Royal Melbourne Hospital, Melbourne, Australia. Published by E. & S. Livingstone, LTD., Edinburgh and London, 1959. Price $12.50.

This book is written as a sequel to one on "Surgery of the Anus, Anal Canal and Rectum," published in 1957. It reflects the author's personal experiences, and from that viewpoint is a worthy addition to the other books on the subject.

The author's concept that clinicians interested in gastrointestinal diseases should not remain divided into physicians and surgeons is interesting, and should be applied in every country. I agree that "management of patients with disorders of the colon will be unsatisfactory until a departure is made from this tradition."

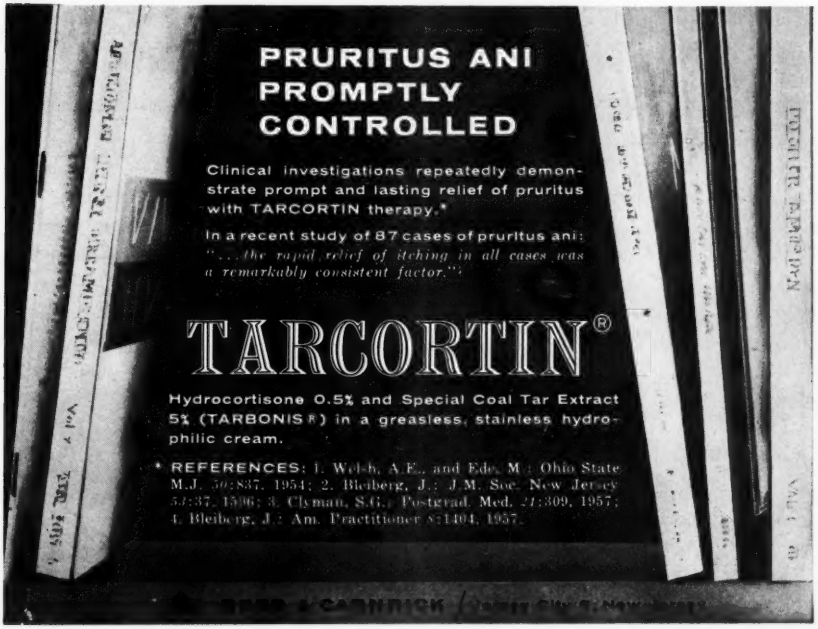
I am therefore also in agreement with the inclusion of chapters on constipation and diarrhea in a surgical textbook.

The book is very well written, and the author's personal viewpoint is evident throughout. Illustrations are very well selected, and surgical techniques are carefully illustrated. This is a book that will be of interest and value to every surgeon.

"A TEXTBOOK OF MEDICINE" edited by Russell L. Cecil, M.D., Sc.D., Professor of Clinical Medicine Emeritus, Cornell University and Robert F. Loeb, M.D., Sc.D., D. Hon. Causa., LI.D., Bard Professor of Medicine, Columbia University. Tenth Edition. Published by W. B. Saunders Company, Philadelphia and London, 1959. Price — about $20.00.

This textbook is a teaching classic. Edited by Russell L. Cecil and Robert F. Loeb, with associate editors, Alexander B. Gutman, Walsh McDermott and Harold G. Wolff, the textbook is one of the finest editions known to this reviewer.

During the past four years there have been many new developments in medicine, and all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BOOK REVIEWS

—Continued from preceding page

are incorporated in the next text. There is new material in many important fields, including adenoviral infections, pharyngoconjunctival fever, cytomegalic inclusion disease, ECHo viral infections, granuloma inguinale, erysipeloid of rosenbach, diseases due to atypical acid-fast bacilli, tropical ulcer, heterodera radicicola, cranial (temporal) arteritis, thrombotic thrombopenic purpura, high altitude sickness, silo Filler's disease, salicylate poisoning, metal fume fever, pantothenic acid deficiency, renal glycosuria, oxalosis, aminoacidurias, renal hypophosphatemia, renal tubular acidosis, agammaglobulinemia, carcinoidosis, hyponatremia and hypokalemia, steroid physiology and metabolism hyperaldosteronism, acute pseudomembranous enterocolitis, hepatic coma, cystic fibrosis of the pancrease, cryoglobulinemia and macroglobulinemia, myeloproliferative disorders, including myeloid metaplasia, cervical spondylosis, benign intracranial hypertension, electric shock treatment in psychiatric therapy, Tietze's syndrome, Marfan's syndrome.

This edition also contains unusually complete index. The book, as always, is well written and beautifully edited. It is very readable and should be in the library of every student and practising physician.

"UROLOGY IN OUTLINE" by T. L. Chapman, Ch.M., F.R.C.S. (England), F.R.F.P.S. (Glas.), Honorary Clinical Lecturer in Urology (University of Glasgow), and Urological Surgeon, Victoria Infirmary, Glasgow; Urological Surgeon, Hairmyres Hospital, Lanarkshire; Urological Surgeon, Ballochmyle Hospital, Ayrshire. Published by the Williams & Wilkins Company, Baltimore. Price $6.75.

This British publication is an excellent outline of an important specialty. It is not intended, of course, for the urologist primarily, but will be particularly useful for the general surgeon and the general practitioner.

The approach to the subject is interesting and different. A series of simple line drawings is used to present even the most complex material, supplemented by very short text content. In this interesting fashion common disease processes and their treatment are illustrated.

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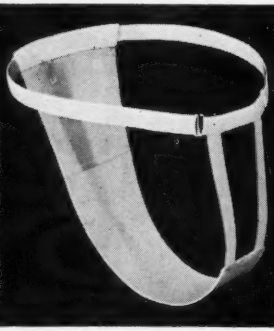

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The Chinese adage on the superiority of pictures over words has been the guiding philosophy of the author in the production of this text.

This reviewer believes that there should be more presentations of this type. The book is recommended particularly for the general practitioner and the general surgeon.

"THE CHEMICAL PREVENTION OF CARDIAC NECROSES" by Hans Selye, M.D., Ph.D., D.Sc., Professor and Director of the Institute of Experimental Medicine and Surgery, University of Montreal, Montreal, Canada. Published by the Ronald Press Company, New York, 1958. Price $7.50.

Hans Selye has obtained unusual eminence as a research authority in stress. He now extends his studies to the chemical prevention of cardiac necroses with exceptionally interesting results.

His text offers a complete synthesis of the experimental observations and clinical studies on this subject. Naturally, it is presented from the viewpoint of the new knowledge on the steroids, stress and electrolytes.

Dr. Selye concludes that treatment with certain electrolytes and steroids will produce cardiac necrosis. Only certain sodium salts will do this after conditioning with steroids.

There may be considerable clinical application to his observation that certain electrolytes will actually prevent the development of such necrosis when they are given simultaneously with steroids.

He also observes that certain steroids of the vitamin-D group can produce such necrosis, perhaps in consequence of tissue calcification.

It is entirely possible that some of the spontaneous cardiac necroses of man may therefore be beneficially influenced by clinical use of common available salts in accordance with Dr. Selye's observations.

If it is true that potassium chloride or magnesium chloride will be of clinical value for man, certainly the contribution in this textbook assumes major proportions. In any case, Dr. Selye's analysis does show a definite interaction between electrolytes and steroids in the production of cardiac necrosis.

Differences in diet (in terms of electrolytes) may be of major importance in relative susceptibility to cardiac necrosis.

Thorough clinical study of this contribution should be made as soon as possible. The text should therefore be carefully studied by the cardiologist, the internist, and the research scientist.

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Actol, The S. E. Massengill Co., Bristol, Tennessee. Liquid, each 5 cc. (teaspoonful) of which contains: 65 mg. neomycin sulfate USP and 5000 units polymyxin B sulfate USP. A bactericide, indicated for use against gram-positive and gram-negative organisms specifically associated with intestinal infections. **Dose:** Adults, 3 or 4 teaspoonfuls three times daily. Children over four years of age, 1 to 2 teaspoonfuls three times daily. Children under four, 1 teaspoonful three times daily. **Sup:** Bottles of 1 pt.

Casakol, The Upjohn Company, Kalamazoo, Michigan. Each capsule or 5 cc. of syrup contains 250 mg. poloxalkol and 30 mg. active constitutent of cascara sagrada. Indicated to provide day-by-day relief of constitpation with virtual absence of side effects. **Dose:** Adults, 1 or 2 capsules, or 1 or 2 teaspoonfuls of syrup at bedtime. Moderately severe constipation, 2 or 3. Children, as directed by physician.

Desitin HC Hemorrhoidal Suppositories, Desitin Chemical Company, Providence, Rhode Island. Each contains 10 mg. hydrocortisone, Norwegian cod liver oil, lanolin, zinc oxide, bismuth subgallate and balsam peru in a cocoa butter base. Indicated to alleviate inflammation, itching, edema, pain and allergic symptoms and to promote healing in severe, acute and chronic inflammatory internal hemorrhoids, proctitis, cryptitis, etc. **Use:** One suppository twice a day up to six days or as required. **Sup:** Boxes of 12.

Glarubin, The S. E. Massengill Company, Bristol, Tennessee. Tablets, each containing 50 mg. glaucarubin (a crystalline glycoside). Indicated for intestinal amebiasis. **Dose:** 3 mg. per kilogram of body weight. 200 mg. is usual adult dose, taken in 2 or 3 equally divided parts for 5 to 10 days. **Sup:** Bottles of 40.

Lavema Enema Powder, Winthrop Laboratories, New York, New York. Packets, each containing 0.02 Gm. oxyphenisatin stabilized with edathamil disodium in lactose q.s. ad 3 Gm. Indicated as a cleansing enema or enema adjuvant. **Use:** As directed by physician. **Sup:** Boxes of 100 packets.

Mucilose-Super Powder, Winthrop Laboratories, New York, New York. A bland hemicellulose obtained from psyllium seed with 50 mg. Superinone per rounded teaspoon. Indicated as a demulcent, bulk laxative and stool softener in chronic atonic or spastic constipation, constipation of pregnancy and rectal constipation and as an adjunct to diets that supply inadequate fecal bulk. **Dose:** As directed by physician. **Sup:** Cans of 4 and 16 oz.

Polykol Capsule, The Upjohn Company, Kalamazoo, Michigan. New dosage form, each capsule containing 250 mg. oxyethylene oxypropylene polymer. Indicated for the prevention and treatment of constipation associated with hard, dry stools. **Dose:** Adults, 2 or 3 capsules daily until regularity is established, then 1 or 2 as needed. Children (6 to 12 years), 1 or 2 capsules daily until regularity is established. **Sup:** Bottles of 16 and 100.

Tral W/Phenobarbital Drops, Abbott Laboratories, North Chicago, Illinois. Mint-chocolate flavor, easily mixed with milk, water, fruit juices, or formula for pediatric use. Indicated for gastrointestinal disorders in infants and children. Also indicated for adults in whom a finer adjustment of the Tral dose or greater ease of administration is desired. **Dose:** As directed by physician. **Sup:** 5-ml. bottle with unbreakable calibrated dropper in packages of 12.

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LETTERS TO THE EDITOR

—Concluded from page 241

fistula surgery, would simply open up avenues for further infection at a time of lowered local and general resistance.

As to the question of prevention of "any" gastrointestinal disturbances, you are quite correct. This is a proofreading error, and should have read many *in place of any. The last sentence in the same paragraph, "not only will this prevent or improve* many *intestinal conditions, but it will help your patients' general health as well,"—will serve to clarify the correct meaning of that paragraph.*

Many thanks for the constructive criticism.

THE EDITOR